```
### Status: Path 1 of [Dialog Information Services via Modem]
 ### Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
 Trying 31060000009999...Open
 DIALOG INFORMATION SERVICES
 PLEASE LOGON:
  ****** HHHHHHHH SSSSSSSS?
 ### Status: Signing onto Dialog
  ****
 ENTER PASSWORD:
  ****** HHHHHHHH SSSSSSS? ******
 Welcome to DIALOG
 ### Status: Connected
 Dialog level 02.05.06D
 Last logoff: 31may02 17:09:18
 Logon file001 04jun02 15:19:39
            *** ANNOUNCEMENT ***
 --Important Notice for Japanese KMKNET Users
 KMKNET will be terminated on 5/31/02. Please
 switch to DLGNET. Please refer to the G-Search
home page at http://www.g-search.or.jp
 for more information.
--SourceOne patents are now delivered to your
email inbox as PDF replacing TIFF delivery.
See HELP SOURCE1 for more information.
--Important news for public and academic
libraries. See HELP LIBRARY for more information.
                    ***
-- Important Notice to Freelance Authors--
See HELP FREELANCE for more information
                    * * *
For information about the access to file 43 please see Help News43.
NEW FILES RELEASED
***AGROProjects (File 235)
***TRADEMARKSCAN-Japan (File 669)
UPDATING RESUMED
***Delphes European Business (File 481)
RELOADED
***CLAIMS/US PATENTS (Files 340, 341, 942)
***Kompass Western Europe (590)
***D&B - Dun's Market Identifiers (516)
REMOVED
***Baton Rouge Advocate (File 382)
***Washington Post (File 146)
***Books in Print (File 470)
***Court Filings (File 793)
***Microcomputer Software Guide Online (File 278)
***Publishers, Distributors & Wholesalers of the U.S. (File 450)
***State Tax Today (File 791)
***Tax Notes Today (File 790)
***Worldwide Tax Daily (File 792)
***New document supplier***
```

IMED has been changed to INFOTRIE (see HELP OINFOTRI)

```
>>>Get immediate news with Dialog's First Release
    news service. First Release updates major newswire
    databases within 15 minutes of transmission over the
    wire. First Release provides full Dialog searchability
    and full-text features. To search First Release files in
    OneSearch simply BEGIN FIRST for coverage from Dialog's
    broad spectrum of news wires.
      >>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
             of new databases, price changes, etc.
 KWIC is set to 50.
 HILIGHT set on as '*'
        1:ERIC 1966-2002/May 10
        (c) format only 2002 The Dialog Corporation
       Set Items Description
           ____
 Cost is in DialUnits
 ?b 155, 5, 73
        04jun02 15:20:18 User259876 Session D352.1
             $0.33 0.095 DialUnits File1
      $0.33 Estimated cost File1
     $0.13 TELNET

$0.46 Estimated cost this search

$0.46 Estimated total session cost 0.095 DialUnits
SYSTEM:OS - DIALOG OneSearch
  File 155:MEDLINE(R)
                       1966-2002/May W4
*File 155: Daily alerts are now available. This file has
been reloaded. Accession numbers have changed.
  File
        5:Biosis Previews(R) 1969-2002/May W4
         (c) 2002 BIOSIS
  File 73:EMBASE 1974-2002/May W4
         (c) 2002 Elsevier Science B.V.
*File 73: For information about Explode feature please
see Help News73.
      Set Items Description
      --- ---- -----
?s (neurogenesis) or (neuronal (w) production)
            8851 NEUROGENESIS
          289734 NEURONAL
         1077895 PRODUCTION
             200 NEURONAL(W) PRODUCTION
            8982 (NEUROGENESIS) OR (NEURONAL (W) PRODUCTION)
?s s1 and ((neurotrophic (w) factor) or (BDNF) or (neurotrophin??))
            8982 S1
           28507 NEUROTROPHIC
                 FACTOR
         1791791
           19787 NEUROTROPHIC (W) FACTOR 7897 BDNF
           13021 NEUROTROPHIN??
      S2
             354 S1 AND ((NEUROTROPHIC (W) FACTOR) OR (BDNF) OR
                  (NEUROTROPHIN??))
?s s2 and (vector or (gene (w) therapy))
Processing
             354 S2
         187121 VECTOR
         1842620 GENE
         4408917 THERAPY
           54023 GENE (W) THERAPY
     S3
              7 S2 AND (VECTOR OR (GENE (W) THERAPY))
```

?rd ...completed examining records 6 RD (unique items) ?t s4/3, k/all

4/3, K/1(Item 1 from file: 5) DIALOG(R) File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.

13340679 BIOSIS NO.: 200100547828

BDNF attenuates ischemia-induced enhancement of *neurogenesis* in rat dentate gyrus.

AUTHOR: Larsson E(a); Mandel R J; Lindvall O(a); Kokaia Z(a) AUTHOR ADDRESS: (a) Section of Restorative Neurology, Wallenberg Neuroscience Center, SE-22184, Lund**Sweden

JOURNAL: Society for Neuroscience Abstracts 27 (2):p1528 2001

MEDIUM: print

CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience

San Diego, California, USA November 10-15, 2001

ISSN: 0190-5295

RECORD TYPE: Abstract LANGUAGE: English

SUMMARY LANGUAGE: English

BDNF attenuates ischemia-induced enhancement of *neurogenesis* in rat dentate gyrus.

ABSTRACT: Brain-derived *neurotrophic* *factor* (*BDNF*) is widely expressed in the adult rodent forebrain and has an important role for neuronal differentiation and survival during embryonic development. To explore the influence of *BDNF* on ischemia-induced cell proliferation and *neurogenesis* in the dentate gyrus of adult rats, the dentate hilus was unilaterally transduced with recombinant adeno-associated virus (rAAV) carrying the *BDNF* and GFP genes. Four to five weeks later the rats were subjected to 30 min of global forebrain ischemia or sham surgery, and proliferating cells were labeled with BrdU during day four to six after the ischemic insult. GFP and *BDNF* immunocytochemistry performed seven weeks after the last BrdU injection showed that rAAV had mainly transduced hilar interneurons and cells in the subgranular layer. Stereological measurement showed increased volume of NPY-immunoreactive cells, indicating that *BDNF* was also biologically active. In the rats transduced with rAAV-GFP only there was a thirteen-fold increase of proliferated neurons. In contrast, in animals transduced with rAAV-*BDNF* we observed decreased number of BrdU-NeuN double-labeled neurons, but no changes of ischemia-induced proliferation, i.e., the number of BrdU-positive cells. We hypothesize that the high levels of *BDNF* attenuated the insult-induced *neurogenesis* by inhibiting the neuronal differentiation of the proliferated hippocampal precursor cells. DESCRIPTORS:

...ORGANISMS: gene *vector*;

CHEMICALS & BIOCHEMICALS: ...brain-derived *neurotrophic* *factor* {

GENE NAME: rat *BDNF* gene (rat brain-derived *neurotrophic* *factor* gene) (Muridae... MISCELLANEOUS TERMS: *neurogenesis*;

4/3,K/2 (Item 2 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.

BIOSIS NO.: 200100519845

Adenoviral infection of the adult rat ventricular zone to overexpress noggin and *BDNF* increases neuronal recruitment from endogenous progenitor cells.

AUTHOR: Chmielnicki E(a); Benraiss A(a); Rosenow J(a); Shore E; Kaplan F;

Economides A N; Goldman S A(a)

AUTHOR ADDRESS: (a) Dept. of Neurology, Cornell U. Med. Col., New York, NY** USA

JOURNAL: Society for Neuroscience Abstracts 27 (1):p939 2001

MEDIUM: print

CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience

San Diego, California, USA November 10-15, 2001

ISSN: 0190-5295

RECORD TYPE: Abstract LANGUAGE: English

SUMMARY LANGUAGE: English

Adenoviral infection of the adult rat ventricular zone to overexpress noggin and *BDNF* increases neuronal recruitment from endogenous progenitor cells.

ABSTRACT: *Neuronal* *production* from ventricular zone (VZ) progenitor cells of the adult rat brain can be induced by neuronal differentiation agents, such as *BDNF* and its adenoviral expression *vector*, AdBDNF. In this study, we asked if *neuronal* *production* by VZ progenitors might also be stimulated by the suppression of glial differentiation using noggin, an inhibitor of the pro-gliogenic BMPs. We also asked if neuronal induction could be further stimulated by concurrent overexpression of *BDNF*. DELTAE1 adenoviruses were made to encode, under CMV control, either DELTAB2 noggin (whose heparin binding site was deleted), or *BDNF* (as an IRES:hGFP). Three groups of 250g rats were injected intraventricularly with either AdNoggin, AdNoggin and AdBDNF together, or an AdGFP control. All 12...

...increase. In each group, >90% of the BrdU+ cells expressed neuronal betaIII-tubulin. Thus, noggin overexpression increased olfactory neuronal recruitment; this effect was accentuated by *BDNF* expression, such that both the suppression of glial and promotion of neuronal differentiation pathways proved viable means for inducing neuronal addition to the adult rat...

DESCRIPTORS:

...ORGANISMS: gene *vector*; ...

...gene *vector*;

CHEMICALS & BIOCHEMICALS: *BDNF* {brain-derived *neurotrophic* *factor* }--

4/3,K/3 (Item 3 from file: 5)

DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.

13291134 BIOSIS NO.: 200100498283

Leukemia inhibitory factor is a key signal for *neurogenesis* in the adult mouse olfactory epithelium.

AUTHOR: Bauer S; Moyse E(a)

AUTHOR ADDRESS: (a) Univ Lyon-1, Villeurbanne**France

JOURNAL: Society for Neuroscience Abstracts 27 (1):p934 2001

MEDIUM: print

CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience

San Diego, California, USA November 10-15, 2001

ISSN: 0190-5295

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

Leukemia inhibitory factor is a key signal for *neurogenesis* in the adult mouse olfactory epithelium.

...ABSTRACT: adulthood. Although culture studies have suggested many candidates, there is no direct evidence from in vivo studies that identifies the molecular signals that control this *neurogenesis*. We

used olfactory bulb ablation (OBX) in adult mice to trigger synchronous and massive mitotic stimulation of neuronal progenitors in olfactory epithelium. Following OBX, we...

- ...in vitro, i.e. leukemia inhibitory factor (LIF), epidermal growth factor, transforming growth factor a, b1, 2, and 3, fibroblast growth factor 2, brain-derived *neurotrophic* *factor*, and tumor necrosis factor a. Of these factors, only LIF is up-regulated before the onset of neuronal progenitor proliferation, and in situ hybridization shows...
- ...mitotic stimulation than in wild type littermates, as measured by bromodeoxyuridine (BrdU) labeling 5 days after OBX. Finally, delivery of exogenous LIF via an adenoviral *vector* to the intact adult olfactory epithelium stimulates BrdU labeling specifically in the olfactory neuron layer. Thus, LIF is a necessary and sufficient inducer of adult olfactory *neurogenesis*.

MISCELLANEOUS TERMS: *neurogenesis*;

4/3,K/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13227416 BIOSIS NO.: 200100434565

Adenoviral brain-derived *neurotrophic* *factor* induces both neostriatal and olfactory neuronal recruitment from endogenous progenitor cells in the adult forebrain.

AUTHOR: Benraiss Abdellatif; Chmielnicki Eva; Lerner Kim; Roh Dongyon; Goldman Steven A(a)

AUTHOR ADDRESS: (a) Department of Neurology and Neuroscience, Cornell University Medical Center, 1300 York Avenue, Room E607, New York, NY, 10021: sgoldm@mail.med.cornell.edu**USA

JOURNAL: Journal of Neuroscience 21 (17):p6718-6731 September 1, 2001 MEDIUM: print

ISSN: 0270-6474

DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English SUMMARY LANGUAGE: English

Adenoviral brain-derived *neurotrophic* *factor* induces both neostriatal and olfactory neuronal recruitment from endogenous progenitor cells in the adult forebrain.

ABSTRACT: Neural progenitor cells persist throughout the adult forebrain subependyma, and neurons generated from them respond to brain-derived *neurotrophic* *factor* (*BDNF*) with enhanced maturation and survival. To induce *neurogenesis* from endogenous progenitors, we overexpressed *BDNF* in the adult ventricular zone by transducing the forebrain ependyma to constitutively express *BDNF*. We constructed a bicistronic adenovirus bearing *BDNF* under cytomegalovirus (CMV) control, and humanized green fluorescent protein (hGFP) under internal ribosomal entry site (IRES) control. This AdCMV:*BDNF*:IRES:hGFP (AdBDNF) was injected into the lateral ventricles of adult rats, who were treated for 18 d thereafter with the mitotic marker bromodeoxyuridine (BrdU). Three weeks after injection, *BDNF* averaged 1 mug/gm in the CSF of AdBDNF-injected animals but was undetectable in control CSF In situ hybridization demonstrated *BDNF* and GFP mRNA expression restricted to the ventricular wall. In AdBDNF-injected rats, the olfactory bulb exhibited a >2.4-fold increase in the number...

...medium spiny neurons of the neostriatum. These newly generated neurons survived at least 5-8 weeks after viral induction, Thus, a single injection of adenoviral *BDNF* substantially augmented the recruitment of new neurons into both neurogenic and non-neurogenic sites in the adult rat brain. The intraventricular delivery of, and ependymal infection by, viral vectors encoding neurotrophic agents may be a feasible strategy for

inducing *neurogenesis* from resident progenitor cells in the adult brain.

DESCRIPTORS:

...ORGANISMS: gene *vector*;

CHEMICALS & BIOCHEMICALS: brain-derived *neurotrophic* *factor*--

...METHODS & EQUIPMENT: *gene* *therapy*--

MISCELLANEOUS TERMS: ...*neurogenesis* induction

4/3,K/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12880787 BIOSIS NO.: 200100087936

Infection of the adult rat ventricular zone by an adenoviral *BDNF*
vector induces neuronal recruitment to the striatum as well as to the
olfactory bulb.

AUTHOR: Chmielnicki E(a); Benraiss A; Lerner K; Roh D; Goldman S A AUTHOR ADDRESS: (a)Cornell Univ. Medical College, New York, NY**USA JOURNAL: Society for Neuroscience Abstracts 26 (1-2):pAbstract No-2083 2000

MEDIUM: print

CONFERENCE/MEETING: 30th Annual Meeting of the Society of Neuroscience New

Orleans, LA, USA November 04-09, 2000

SPONSOR: Society for Neuroscience

ISSN: 0190-5295

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

Infection of the adult rat ventricular zone by an adenoviral *BDNF* *vector* induces neuronal recruitment to the striatum as well as to the olfactory bulb.

ABSTRACT: Neural progenitor cells persist in the forebrain subependyma, and neurons generated from them respond to brain-derived *neurotrophic* *factor* (*BDNF*) with enhanced maturation and survival. To induce *neurogenesis* from resident progenitors, we transduced the adult ventricular wall to overexpress *BDNF*. We constructed a DELTAE1 adenovirus bearing *BDNF* under CMV promoter control, in tandem with hGFP under IRES control. This AdCMV:*bdnf*:IRES:hgfp was injected into the lateral ventricles of adult rats, which were then injected intraperitoneally for 18 d with the mitotic marker BrdU (150 mug/g/d). 3 weeks after virus injection, the CSF concentration of *BDNF* was >1 ng/mg protein in AdBDNF-injected animals, but undetectable in control CSF. In situ hybridization revealed high-level *BDNF* and GFP mRNA expression in the AdBDNF-treated ventricular walls. Among AdBDNF-injected rats, the olfactory bulb harbored >2.5-fold the number of BrdU...

...tubulin+ striatal neurons were typically found in clusters, that were found almost exclusively in the AdBDNF-treated rats. Thus, a single injection of an adenoviral *BDNF* *vector* substantially augmented neuronal recruitment into the adult rat brain. The AdBDNF-associated neurons integrated not only into the normally neurogenic olfactory bulb, but also into the otherwise non-neurogenic neostriatum. As such, the intraventricular delivery of viral vectors encoding *neurotrophins* is a feasible strategy for inducing *neurogenesis* from resident neural progenitor cells. This may have particular implications in diseases of neostriatal degeneration, such as Huntington's disease.

CHEMICALS & BIOCHEMICALS: *BDNF* {brain-derived *neurotrophic* *factor* }; ...

...adenoviral *BDNF* *vector* {adenoviral brain-derived *neurotrophic*
 factor *vector*}-MISCELLANEOUS TERMS: *neurogenesis*;

```
4/3,K/6
              (Item 6 from file: 5)
 DIALOG(R)File
                 5:Biosis Previews(R)
 (c) 2002 BIOSIS. All rts. reserv.
 12391389 BIOSIS NO.: 200000144891
 In yavo transduction of the adult rat ventricular zone with an adenoviral
   *BDNF* *vector* increases *neuronal* *production* and recruitment to the
   olfactory bulb.
 AUTHOR: Benraiss A(a); Lerner K(a); Chmielnicki E(a); Hackett N; Crystal R
   G; Goldman S A(a)
 AUTHOR ADDRESS: (a) Dept. of Neurology and Neuroscience, Cornell Univ.
   Medical College, New York, NY, 10021**USA
 JOURNAL: Society for Neuroscience Abstracts. 25 (1-2):p1028 1999
 CONFERENCE/MEETING: 29th Annual Meeting of the Society for Neuroscience.
 Miami Beach, Florida, USA October 23-28, 1999
 SPONSOR: Society for Neuroscience
 ISSN: 0190-5295
 RECORD TYPE: Citation
 LANGUAGE: English
 SUMMARY LANGUAGE: English
 In vivo transduction of the adult rat ventricular zone with an adenoviral
  *BDNF* *vector* increases *neuronal* *production* and recruitment to the
  olfactory bulb.
DESCRIPTORS:
   ...ORGANISMS: *vector*;
  CHEMICALS & BIOCHEMICALS:
                               brain-derived *neurotrophic* *factor*--
  MISCELLANEOUS TERMS: *neuronal* *production*;
?ds
Set
        Items
                Description
         8982
                 (NEUROGENESIS) OR (NEURONAL (W) PRODUCTION)
S2
          354
                S1 AND ((NEUROTROPHIC (W) FACTOR) OR (BDNF) OR (NEUROTROPH-
             IN??))
S3
            7
                S2 AND (VECTOR OR (GENE (W) THERAPY))
S4
            6
                RD (unique items)
?s s2 and (vector)
             354 52
          187121 VECTOR
               7 S2 AND (VECTOR)
?s s2 and (treatment or therapy)
             354 S2
         3813291 TREATMENT
         4408917 THERAPY
              40 S2 AND (TREATMENT OR THERAPY)
?s s6 and (neurodegenerative (w) condition)
              40 S6
           25650 NEURODEGENERATIVE
          373846 CONDITION
                  NEURODEGENERATIVE(W)CONDITION
             144
               0 S6 AND (NEURODEGENERATIVE (W) CONDITION)
?s s6 and (Huntington's (w) disease)
>>>Warning: unmatched quote found
              40
                 S6
                  HUNTINGTON'S
               0
         4449708
                  DISEASE
                  HUNTINGTON'S(W)DISEASE
      S8
               0
                 S6 AND (HUNTINGTON'S (W) DISEASE)
?s s6 and (Huntington)
              40 S6
           17544 HUNTINGTON
     S9
              2 S6 AND (HUNTINGTON)
...completed examining records
    S10
              2 RD (unique items)
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10/3,K/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

13227416 BIOSIS NO.: 200100434565

Adenoviral brain-derived *neurotrophic* *factor* induces both neostriatal and olfactory neuronal recruitment from endogenous progenitor cells in the adult forebrain.

AUTHOR: Benraiss Abdellatif; Chmielnicki Eva; Lerner Kim; Roh Dongyon; Goldman Steven A(a)

AUTHOR ADDRESS: (a) Department of Neurology and Neuroscience, Cornell University Medical Center, 1300 York Avenue, Room E607, New York, NY, 10021: sgoldm@mail.med.cornell.edu**USA

JOURNAL: Journal of Neuroscience 21 (17):p6718-6731 September 1, 2001

MEDIUM: print ISSN: 0270-6474

DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English

SUMMARY LANGUAGE: English

Adenoviral brain-derived *neurotrophic* *factor* induces both neostriatal and olfactory neuronal recruitment from endogenous progenitor cells in the adult forebrain.

ABSTRACT: Neural progenitor cells persist throughout the adult forebrain subependyma, and neurons generated from them respond to brain-derived *neurotrophic* *factor* (*BDNF*) with enhanced maturation and survival. To induce *neurogenesis* from endogenous progenitors, we overexpressed *BDNF* in the adult ventricular zone by transducing the forebrain ependyma to constitutively express *BDNF*. We constructed a bicistronic adenovirus bearing *BDNF* under cytomegalovirus (CMV) control, and humanized green fluorescent protein (hGFP) under internal ribosomal entry site (IRES) control. This AdCMV:*BDNF*:IRES:hGFP (AdBDNF) was injected into the lateral ventricles of adult rats, who were treated for 18 d thereafter with the mitotic marker bromodeoxyuridine (BrdU). Three weeks after injection, *BDNF* averaged 1 mug/gm in the CSF of AdBDNF-injected animals but was undetectable in control CSF In situ hybridization demonstrated *BDNF* and GFP mRNA expression restricted to the ventricular wall. In AdBDNF-injected rats, the olfactory bulb exhibited a >2.4-fold increase in the number...

- ...tubulin+ neurons, confirmed by confocal imaging, relative to AdNull (AdCMV:hGFP) controls. Importantly, AdBDNF-associated neuronal recruitment to the neostriatum was also noted, with the *treatment* -induced addition of BrdU+-NeuN+-betaIII-tubulin+ neurons to the caudate putamen. Many of these cells also expressed glutamic acid decarboxylase, cabindin-D28, and DARPP...
- ...medium spiny neurons of the neostriatum. These newly generated neurons survived at least 5-8 weeks after viral induction, Thus, a single injection of adenoviral *BDNF* substantially augmented the recruitment of new neurons into both neurogenic and non-neurogenic sites in the adult rat brain. The intraventricular delivery of, and ependymal infection by, viral vectors encoding neurotrophic agents may be a feasible strategy for inducing *neurogenesis* from resident progenitor cells in the adult brain.

DESCRIPTORS:

DISEASES: *Huntington*'s disease...

CHEMICALS & BIOCHEMICALS: brain-derived *neurotrophic* *factor*--

...METHODS & EQUIPMENT: gene *therapy*--

MISCELLANEOUS TERMS: ...*neurogenesis* induction ALTERNATE INDEXING: *Huntington*'s Disease (MeSH)

```
DIALOG(R)File
                73:EMBASE
 (c) 2002 Elsevier Science B.V. All rts. reserv.
 10939981
              EMBASE No: 2000430209
   Neuroprotective signaling and the aging brain: Take away my food and let
 me run
   Mattson M.P.
  M.P. Mattson, Laboratory of Neurosciences, National Institute Aging,
   Gerontology Research Center, 5600 Nathan Shock Drive, Baltimore, MD
   21224-6825 United States
   AUTHOR EMAIL: mattsonm@grc.nia.nih.gov
   Brain Research (BRAIN RES.) (Netherlands) 15 DEC 2000, 886/1-2 (47-53)
   CODEN: BRREA
                  ISSN: 0006-8993
   PUBLISHER ITEM IDENTIFIER: S0006899300027906
   DOCUMENT TYPE: Journal; Review
   LANGUAGE: ENGLISH
                     SUMMARY LANGUAGE: ENGLISH
   NUMBER OF REFERENCES: 51
   ...calorie intake) can increase the resistance of neurons in the brain to
 dysfunction and death in experimental models of Alzheimer's disease,
Parkinson's disease, *Huntington*'s disease and stroke. The mechanism
underlying the beneficial effects of dietary restriction involves
stimulation of the expression of 'stress proteins' and neurotrophic
 factors. The...
 ... manipulation can increase the brain's capacity for plasticity and
self-repair. Work in other laboratories suggests that physical and
intellectual activity can similarly increase *neurotrophic* *factor*
production and *neurogenesis*. Collectively, the available data suggest the
that dietary restriction, and physical and mental activity, may reduce both
the incidence and severity of neurodegenerative disorders in...
DRUG DESCRIPTORS:
**neurotrophic* *factor*--endogenous compound--ec
MEDICAL DESCRIPTORS:
signal transduction; brain; food intake; nerve cell; cell survival; nerve
cell plasticity; degenerative disease--*therapy*--th; degenerative disease
--prevention--pc; degenerative disease--etiology--et; degenerative disease
--epidemiology--ep; exercise; risk factor; hyperphagia; cardiovascular
disease--etiology--et; diabetes mellitus--etiology--et; cancer--etiology
--et; caloric intake; Alzheimer disease--etiology--et; Parkinson disease
--etiology--et; *Huntington* chorea--etiology--et; stroke--etiology--et;
calcium homeostasis; calcium cell level; apoptosis; cell regeneration;
nervous system development; physical activity; mental performance;
mitochondrion; human; nonhuman; review...
?ds
Set
        Items
                Description
S1
         8982
                (NEUROGENESIS) OR (NEURONAL (W) PRODUCTION)
S2
          354
                S1 AND ((NEUROTROPHIC (W) FACTOR) OR (BDNF) OR (NEUROTROPH-
             IN??))
S3
            7
                S2 AND (VECTOR OR (GENE (W) THERAPY))
S4
                RD (unique items)
            6
S5
            7
                S2 AND (VECTOR)
56
           40
                S2 AND (TREATMENT OR THERAPY)
S7
           0
                S6 AND (NEURODEGENERATIVE (W) CONDITION)
S8
           0
                S6 AND (HUNTINGTON'S (W) DISEASE)
S9
                S6 AND (HUNTINGTON)
           2
S10
                RD (unique items)
?rd s6
...completed examining records
     S11
             27 RD S6 (unique items)
?s s11 and review
              27 S11
         1243606 REVIEW
```

10/3, K/2

S12

?t s12/3, k/all

4 S11 AND REVIEW

(Item 1 from file: 73)

12/3,K/1 (Item 1 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

10929592 20496991 PMID: 11040417

Neuroprotection by estradiol.

Garcia-Segura L M; Azcoitia I; DonCarlos L L

Instituto Cajal, CSIC, Madrid, Spain.

Progress in neurobiology (ENGLAND) Jan 2001, 63 (1) p29-60, ISSN 0301-0082 Journal Code: 0370121

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

This *review* highlights recent evidence from clinical and basic science studies supporting a role for estrogen in neuroprotection. Accumulated clinical evidence suggests that estrogen exposure decreases the...

... this protective role through several routes. Key among these are estrogen dependent alterations in cell survival, axonal sprouting, regenerative responses, enhanced synaptic transmission and enhanced *neurogenesis*. Some of the mechanisms underlying these effects are independent of the classically defined nuclear estrogen receptors and unidentified membrane receptors, direct modulation involve neurotransmitter...

... confer resistance to injury. Although there is clear evidence that estradiol exposure can be deleterious to some neuronal populations, the potential clinical benefits of estrogen *treatment* for enhancing cognitive function may outweigh the associated central and peripheral risks. Exciting and important avenues for future investigation into the protective effects of estrogen include the optimal ligand and doses that can be used clinically to confer benefit without undue risk, modulation of *neurotrophin* and *neurotrophin* receptor expression, interaction of estrogen with regulated cofactors and coactivators that couple estrogen receptors to basal transcriptional machinery, interactions of estrogen with other survival and...

12/3, K/2(Item 1 from file: 73)

DIALOG(R)File 73:EMBASE

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11441688 EMBASE No: 2002013750

Regulation of adult *neurogenesis* by antidepressant *treatment*

Duman R.S.; Nakagawa S.; Malberg J.

Dr. R.S. Duman, Abraham Ribicoff Research Facilities, Yale University School of Medicine, 34 Park Street, New Haven, CT 06508 United States AUTHOR EMAIL: ronald.duman@yale.edu

Neuropsychopharmacology (NEUROPSYCHOPHARMACOLOGY) (United States)

2001, 25/6 (836-844)

CODEN: NEROE ISSN: 0893-133X

PUBLISHER ITEM IDENTIFIER: S0893133X0100358X

DOCUMENT TYPE: Journal ; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 42

Regulation of adult *neurogenesis* by antidepressant *treatment*

Demonstration of *neurogenesis* in adult brain represents a major advance in our understanding of the cellular mechanisms underlying neuronal remodeling and complex behavior. Recent studies from our laboratory and others demonstrate that chronic administration of an antidepressant, including either a 5-HT or norepinephrine selective reuptake inhibitor, up-regulates *neurogenesis* in adult rodent hippocampus. Up-regulation of

neurogenesis could block or reverse the effects of stress on hippocampal neurons, which include down-regulation of *neurogenesis*, as well as atrophy. The possibility that the cAMP signal transduction cascade contributes to the regulation of *neurogenesis* by antidepressants is supported by previous studies and by recent work. Although additional studies must be conducted to determine the significance of adult *neurogenesis* in humans, these findings will stimulate new avenues of research to identify the cellular and molecular basis of stress-related mood disorders as well as... DRUG DESCRIPTORS:

cyclic AMP--endogenous compound--ec; monoamine oxidase inhibitor --pharmacology--pd; lithium--pharmacology--pd; morphine--pharmacology--pd; haloperidol--pharmacology--pd; brain derived *neurotrophic* *factor* --endogenous compound--ec; *neurotrophic* *factor*--endogenous compound--ec ; serotonin--endogenous compound--ec; cyclic AMP responsive element binding protein--endogenous compound--ec; rolipram--pharmacology--pd MEDICAL DESCRIPTORS:

...region; stress; down regulation; atrophy; signal transduction; long term exposure; nerve cell; affective neurosis--etiology--et; neuropathology; cell maturation; protein localization; protein expression; human; nonhuman; *review*; priority journal

...CAS REGISTRY NO.: 57-27-2 (morphine); 52-86-8 (haloperidol); 218441-99-7 (brain derived *neurotrophic* *factor*); 50-67-9 (serotonin); 130428-87-4...

12/3,K/3 (Item 2 from file: 73) DIALOG(R)File 73:EMBASE (c) 2002 Elsevier Science B.V. All rts. reserv.

10939981 EMBASE No: 2000430209

Neuroprotective signaling and the aging brain: Take away my food and let me run

Mattson M.P.

M.P. Mattson, Laboratory of Neurosciences, National Institute Aging, Gerontology Research Center, 5600 Nathan Shock Drive, Baltimore, MD 21224-6825 United States

AUTHOR EMAIL: mattsonm@grc.nia.nih.gov

Brain Research (BRAIN RES.) (Netherlands) 15 DEC 2000, 886/1-2 (47-53)

CODEN: BRREA ISSN: 0006-8993

PUBLISHER ITEM IDENTIFIER: S0006899300027906

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 51

... manipulation can increase the brain's capacity for plasticity and self-repair. Work in other laboratories suggests that physical and intellectual activity can similarly increase *neurotrophic* *factor* production and *neurogenesis*. Collectively, the available data suggest the that dietary restriction, and physical and mental activity, may reduce both the incidence and severity of neurodegenerative disorders in... DRUG DESCRIPTORS:

**neurotrophic* *factor*--endogenous compound--ec MEDICAL DESCRIPTORS:

signal transduction; brain; food intake; nerve cell; cell survival; nerve cell plasticity; degenerative disease--*therapy*--th; degenerative disease --prevention--pc; degenerative disease--etiology--et; degenerative disease --epidemiology--ep; exercise; risk factor; hyperphagia; cardiovascular disease--etiology--et; diabetes mellitus--etiology--et...

...Huntington chorea--etiology--et; stroke--etiology--et; calcium homeostasis; calcium cell level; apoptosis; cell regeneration; nervous system development; physical activity; mental performance; mitochondrion; human; nonhuman; *review*; priority journal

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12/3,K/4
              (Item 3 from file: 73)
DIALOG(R) File 73: EMBASE
(c) 2002 Elsevier Science B.V. All rts. reserv.
10611471
             EMBASE No: 2000077541
 Neural plasticity to stress and antidepressant *treatment*
 Duman R.S.; Malberg J.; Thome J.
 Dr. R.S. Duman, 34 Park Street, New Haven, CT 06508 United States
 Biological Psychiatry ( BIOL. PSYCHIATRY ) (United States) 01 NOV 1999,
 46/9 (1181-1191)
 CODEN: BIPCB
                ISSN: 0006-3223
 PUBLISHER ITEM IDENTIFIER: S0006322399001778
 DOCUMENT TYPE: Journal; Review
 LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
 NUMBER OF REFERENCES: 55
```

Neural plasticity to stress and antidepressant *treatment*

Adaptations at the cellular and molecular levels in response to stress and antidepressant *treatment* could represent a form of neural plasticity that contributes to the pathophysiology and *treatment* of depression. At the cellular level, atrophy and death of stress-vulnerable neurons in the hippocampus, as well as decreased *neurogenesis* of hippocampal neurons, has been reported in preclinical studies. Clinical studies also provide evidence for atrophy and cell death in the hippocampus, as well as the prefrontal cortex. It is possible that antidepressant *treatment* could oppose these adverse cellular effects, which may be regarded as a loss of neural plasticity, by blocking or reversing the atrophy of hippocampal neurons...

DRUG DESCRIPTORS:

cyclic AMP--endogenous compound--ec; *neurotrophic* *factor*--endogenous compound--ec; glutamic acid--endogenous compound--ec; brain derived *neurotrophic* *factor*--endogenous compound--ec; tianeptine--pharmacology --pd; cyclic AMP responsive element binding protein--endogenous compound --ec; beta adrenergic receptor--endogenous compound--ec; mitogen activated protein knase...

MEDICAL DESCRIPTORS:

24 S14

hippocampus; prefrontal cortex; cell death; atrophy; signal transduction; dentate gyrus; brain atrophy; glucose transport; protein expression; cell proliferation; cell differentiation; gene expression regulation; human; nonhuman; *review*; priority journal?ds

```
Set
        Items
                Description
S1
         8982
                 (NEUROGENESIS) OR (NEURONAL (W) PRODUCTION)
S2
          354
                S1 AND ((NEUROTROPHIC (W) FACTOR) OR (BDNF) OR (NEUROTROPH-
             IN??))
S3
            7
                S2 AND (VECTOR OR (GENE (W) THERAPY))
S4
            6
                RD (unique items)
S5
            7
                S2 AND (VECTOR)
S6
                S2 AND (TREATMENT OR THERAPY)
           40
S7
                S6 AND (NEURODEGENERATIVE (W) CONDITION)
            0
S8
            0
                S6 AND (HUNTINGTON'S (W) DISEASE)
S9
            2
                S6 AND (HUNTINGTON)
S10
            2
                RD (unique items)
S11
           27
                RD S6 (unique items)
S12
            4
                S11 AND REVIEW
?s s2 and review
             354
                 S2
         1243606 REVIEW
              28 S2 AND REVIEW
...completed examining records
     S14
              24 RD (unique items)
?s s14 and (Huntington)
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15/3,K/1 (Item 1 from file: 73) DIALOG(R) File 73: EMBASE

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10939981 EMBASE No: 2000430209

Neuroprotective signaling and the aging brain: Take away my food and let

Mattson M.P.

S15

M.P. Mattson, Laboratory of Neurosciences, National Institute Aging, Gerontology Research Center, 5600 Nathan Shock Drive, Baltimore, MD 21224-6825 United States

AUTHOR EMAIL: mattsonm@grc.nia.nih.gov

Brain Research (BRAIN RES.) (Netherlands) 15 DEC 2000, 886/1-2 (47-53)

CODEN: BRREA ISSN: 0006-8993

PUBLISHER ITEM IDENTIFIER: S0006899300027906

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 51

...calorie intake) can increase the resistance of neurons in the brain to dysfunction and death in experimental models of Alzheimer's disease, Parkinson's disease, *Huntington*'s disease and stroke. The mechanism underlying the beneficial effects of dietary restriction involves stimulation of the expression of 'stress proteins' and neurotrophic factors. The...

... manipulation can increase the brain's capacity for plasticity and self-repair. Work in other laboratories suggests that physical and intellectual activity can similarly increase *neurotrophic* *factor* production and *neurogenesis*. Collectively, the available data suggest the that dietary restriction, and physical and mental activity, may reduce both the incidence and severity of neurodegenerative disorders in... DRUG DESCRIPTORS:

**neurotrophic* *factor*--endogenous compound--ec MEDICAL DESCRIPTORS:

...exercise; risk factor; hyperphagia; cardiovascular disease--etiology--et ; diabetes mellitus--etiology--et; cancer--etiology--et; caloric intake; Alzheimer disease--etiology--et; Parkinson disease--etiology--et; *Huntington* chorea--etiology--et; stroke--etiology--et; calcium homeostasis; calcium cell level; apoptosis; cell regeneration; nervous system development; physical activity; mental performance; mitochondrion; human; nonhuman; *review*; priority journal ?ds

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Set
        Items
                Description
S1
         8982
                (NEUROGENESIS) OR (NEURONAL (W) PRODUCTION)
S2
                S1 AND ((NEUROTROPHIC (W) FACTOR) OR (BDNF) OR (NEUROTROPH-
          354
             IN??))
S3
            7
                S2 AND (VECTOR OR (GENE (W) THERAPY))
S4
                RD (unique items)
            6
S5
            7
                S2 AND (VECTOR)
S6
           40
                S2 AND (TREATMENT OR THERAPY)
s7
            0
                S6 AND (NEURODEGENERATIVE (W) CONDITION)
S8
            0
                S6 AND (HUNTINGTON'S (W) DISEASE)
S9
            2
                S6 AND (HUNTINGTON)
S10
           2
                RD (unique items)
S11
           27
                RD S6 (unique items)
S12
           4
                S11 AND REVIEW
S13
           28
                S2 AND REVIEW
S14
           24
                RD (unique items)
S15
           1
                S14 AND (HUNTINGTON)
?t s14/3,k/all
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14/3,K/1 (Item 1 from file: 155) DIALOG(R) File 155: MEDLINE(R)

11060724 21064491 PMID: 11124444

The role of cytokines and growth factors in seizures and their sequelae.

Jankowsky J L; Patterson P H

Biology Division, California Institute of Technology, 216-76 Caltech, Pasadena, CA 91125, USA.

Progress in neurobiology (England) Feb 2001, 63 (2) p125-49, ISSN 0301-0082 Journal Code: 0370121

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

... caused by severe or repeated seizures have been well characterized, many questions about the molecular mechanisms involved remain unanswered. Neuronal cell death, reactive gliosis, enhanced *neurogenesis*, and axonal sprouting are four of the best-studied sequelae of seizures. In vitro, each of these pathological processes can be substantially influenced by soluble protein factors, including *neurotrophins*, cytokines, and growth factors. Furthermore, many of these proteins and their receptors are expressed in the adult brain and are up-regulated in response to neuronal activity and injury. We *review* the evidence that these intercellular signaling proteins regulate seizure activity as well as subsequent pathology in vivo. As nerve growth factor and brain derived *neurotrophic* *factor* are the best-studied proteins of this class, we begin by discussing the evidence linking these *neurotrophins* to epilepsy and seizure. More than a dozen additional cytokines, growth factors, and *neurotrophins* that have been examined in the context of epilepsy models are then considered. We discuss the effect of seizure on expression of cytokines and growth...

; Astrocytes--metabolism--ME; Astrocytes--pathology--PA; Brain-Derived *Neurotrophic* *Factor*--genetics--GE; Brain-Derived *Neurotrophic* *Factor*--metabolism--ME; Cell Death; Cytokines--genetics--GE; Disease Models, Animal; Growth Substances--genetics--GE; Microglia--metabolism--ME; Microglia--pathology--PA; Mossy Fibers, Hippocampal--pathology--PA...

Chemical Name: Brain-Derived *Neurotrophic* *Factor*; Cytokines; Growth Substances; Nerve Growth Factors; RNA, Messenger

14/3,K/2 (Item 2 from file: 155) DIALOG(R) File 155: MEDLINE(R)

20496991 PMID: 11040417

Neuroprotection by estradiol. Garcia-Segura L M; Azcoitia I; DonCarlos L L

Instituto Cajal, CSIC, Madrid, Spain.

Progress in neurobiology (ENGLAND) Jan 2001, 63 (1) p29-60, ISSN 0301-0082 Journal Code: 0370121

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

This *review* highlights recent evidence from clinical and basic science studies supporting a role for estrogen in neuroprotection. Accumulated clinical evidence suggests that estrogen exposure decreases the...

... this protective role through several routes. Key among these are estrogen dependent alterations in cell survival, axonal sprouting, regenerative responses, enhanced synaptic transmission and enhanced *neurogenesis* . Some of the mechanisms underlying these effects are independent of the classically defined nuclear estrogen receptors and unidentified membrane receptors, direct modulation of involve neurotransmitter...

... into the protective effects of estrogen include the optimal ligand and doses that can be used clinically to confer benefit without undue risk, modulation of *neurotrophin* and *neurotrophin* receptor expression, interaction of estrogen with regulated cofactors and coactivators that couple estrogen receptors to basal transcriptional machinery, interactions of estrogen with other survival and...

14/3,K/3 (Item 3 from file: 155) DIALOG(R)File 155:MEDLINE(R)

10811752 20351308 PMID: 10891876

Neurotrophins and development of the rod pathway: an elementary deduction.

Rickman D W

Department of Ophthalmology and Visual Sciences, University of Iowa College of Medicine, Iowa City, Iowa. rickm004@mc.duke.edu

Microscopy research and technique (UNITED STATES) Jul 15 2000, 50 (2) p124-9, ISSN 1059-910X Journal Code: 9203012

Contract/Grant No.: EY11389; EY; NEI

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

Neurotrophins and development of the rod pathway: an elementary deduction.

The rodent retina is a particularly attractive model for the study of neuronal developmental processes since considerable *neurogenesis*, cellular migration, phenotypic differentiation of retinal cell types and synaptogenesis occurs postnatally. In addition, the retina is readily accessible to surgical intervention, pharmacological manipulation, and... ... expression—tools that can be utilized to study mechanisms underlying the development of retinal neurons and their interconnections that form distinct functional circuits. Here, I *review* our studies describing the ontogeny of a specific retinal interneuron, the AII amacrine cell, an integral element in the rod (scotopic) pathway. Specifically, we used...

; Brain-Derived *Neurotrophic* *Factor*--genetics--GE; Brain-Derived *Neurotrophic* *Factor*--metabolism--ME; Immunohistochemistry; In Situ Hybridization; Interneurons--metabolism--ME; Nerve Growth Factor--genetics--GE; Nerve Growth Factor--metabolism--ME; Oligonucleotides, Antisense --pharmacology--PD; Parvalbumins--analysis...

Chemical Name: Brain-Derived *Neurotrophic* *Factor*; Nerve Growth Factors; Oligonucleotides, Antisense; Parvalbumins; RNA, Messenger; Thionucleotides; Nerve Growth Factor; Receptor, trkB

14/3,K/4 (Item 4 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

09809893 98248177 PMID: 9588763

Neurotrophic factors and the maldevelopmental hypothesis of schizophrenic psychoses. *Review* article.

Thome J; Foley P; Riederer P

Department of Psychiatry, University of Wurzburg, Federal Republic of Germany.

Journal of neural transmission (Vienna, Austria: 1996) (AUSTRIA) 1998, 105 (1) p85-100, ISSN 0300-9564 Journal Code: 9702341

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

Neurotrophic factors and the maldevelopmental hypothesis of schizophrenic psychoses. *Review* article.

The maldevelopmental model of schizophrenia postulates pathological

alterations in embryonal *neurogenesis* as the etiopathogenetic basis of schizophrenic psychosis; the *neurotrophic* *factor* hypothesis explains these changes as the result of disturbances of processes involving the trophic factors. Neurotransmitter deficits are thereby interpreted as epiphenomena of underlying *neurotrophic* *factor* deficacy. The functional systems of the various neurotrophic factors are characterized by complex interaction mechanisms. Both primary genetic alterations, and secondary impairments, induced by exogene...

... be associated with changes in the genetic code of certain neurotrophic factors. Various phenomena typical of the schizophrenic psychoses can be interpreted according to the *neurotrophic* *factor* hypothesis.

14/3,K/5 (Item 5 from file: 155) DIALOG(R) File 155:MEDLINE(R)

09308552 97209422 PMID: 9056722

Developmental plasticity in neural circuits for a learned behavior.

Bottjer S W; Arnold A P

Department of Biology, University of Southern California, Los Angeles 90089-2520, USA.

Annual review of neuroscience (UNITED STATES) 1997, 20 p459-81,

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH Main Citation Owner: NLM Record type: Completed

...both neural and behavioral change during development and has taught us valuable information regarding sensitive periods, rearrangement of synaptic connections, topographic specificity, cell death and *neurogenesis*, experience-dependent neural plasticity, and sexual differentiation. The song system differs in some interesting ways from some well-studied mammalian model systems and thus offers fresh perspectives on specific theoretical issues. In this highly selective *review*, we concentrate on two major questions: What are the developmental changes in the song system responsible for song learning and the restriction of learning to... ... sensitive period, and what factors explain the highly sexually dimorphic development of this system? We discuss the important role of sex steroid hormones and of *neurotrophins* in creating a male-typical neural song circuit (which can learn to produce complex vocalizations) instead of a reduced, female-typical song circuit that does...

14/3,K/6 (Item 1 from file: 5) DIALOG(R) File 5: Biosis Previews (R) (c) 2002 BIOSIS. All rts. reserv.

07442345 BIOSIS NO.: 000040033659

THE ROLE OF GROWTH FACTORS IN THE CONTROL OF *NEUROGENESIS*

AUTHOR: ROHRER H

AUTHOR ADDRESS: MAX-PLANCK-INST. PSYCHIATRIE, NEUROCHEM., 8033 MARTINSRIED/PLANEGG, AM KLOPFERSPITZ 18A, FRG.

JOURNAL: EUR J NEUROSCI 2 (12). 1990. 1005-1015. 1990

CODEN: EJONE

DOCUMENT TYPE: Review RECORD TYPE: Citation LANGUAGE: ENGLISH

THE ROLE OF GROWTH FACTORS IN THE CONTROL OF *NEUROGENESIS* DESCRIPTORS: *REVIEW* CHICK RAT MOUSE VERTEBRATE IN-VITRO STUDIES CILIARY *NEUROTROPHIC* *FACTOR* INSULIN FIBROBLAST GROWTH FACTOR INSULIN-LIKE GROWTH FACTOR NERVE GROWTH FACTOR NEURONAL PROLIFERATION NEURONAL DIFFERENTIATION CENTRAL NERVOUS SYSTEM PERIPHERAL NERVOUS SYSTEM

14/3,K/7 (Item 1 from file: 73)

DIALOG(R) File 73: EMBASE

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11586560 EMBASE No: 2002158257

The common properties of *neurogenesis* in the adult brain: From invertebrates to vertebrates

Cayre M.; Malaterre J.; Scotto-Lomassese S.; Strambi C.; Strambi A. M. Cayre, CNRS, Laboratoire de Neurobiologie, 31 Chemin Joseph Aiguier, 13402 Marseille Cedex 20 France

AUTHOR EMALL: cayre@ibdm.univ-mrs.fr

Comparative Biochemistry and Physiology - B Biochemistry and Molecular Biology (COMP. BIOCHEM. PHYSIOL. B BIOCHEM. MOL. BIOL.) (United States)

adones

2002, 132/1 (1-15)

CODEN: CBPBB ISSN: 1096-4959

PUBLISHER ITEM IDENTIFIER: S1096495901005255

DOCUMENT TYPE: Journal ; Conference Paper

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 156

The common properties of *neurogenesis* in the adult brain: From invertebrates to vertebrates

...nervous system. In vertebrates, the subventricular zone and the dentate gyrus of the hippocampus are the sites of neuronal precursor proliferation. In some insects, persistent *neurogenesis* occurs in the mushroom bodies, which are brain structures involved in learning and memory and considered as functional analogues of the hippocampus. In both vertebrates and invertebrates, secondary *neurogenesis* (including neuroblast proliferation and neuron differentiation) appears to be regulated by hormones, transmitters, growth factors and environmental cues. The functional implications of adult *neurogenesis* have not yet been clearly demonstrated and comparative study of the various model systems could contribute to better understand this phenomenon. Here, we *review* and discuss the common characteristics of adult *neurogenesis* in the various animal models studied so far. (c) 2002 Elsevier Science Inc. All rights reserved.

DRUG DESCRIPTORS:

...2--endogenous compound--ec; epidermal growth factor--endogenous compound

--ec; somatomedin C--endogenous compound--ec; nerve growth factor

--endogenous compound--ec; glial cell line derived *neurotrophic* *factor*

--endogenous compound--ec; brain derived *neurotrophic* *factor*

--endogenous compound--ec; cyclic AMP responsive element binding protein binding protein-endogenous compound--ec; unclassified drug

...CAS REGISTRY NO.: 9 (nitric oxide); 62229-50-9 (epidermal growth factor); 67763-96-6 (somatomedin C); 9061-61-4 (nerve growth factor); 218441-99-7 (brain derived *neurotrophic* *factor*); 190209-80-4 (cyclic AMP responsive element binding protein binding protein)

14/3,K/8 (Item 2 from file: 73)

DIALOG(R)File 73:EMBASE

(c) 2002 Elsevier Science B.V. All rts. reserv.

11500226 EMBASE No: 2002071866

Nervous primary cells in the adult: Recent progress and contribution from the olfactory system

LES CELLULES-SOUCHES NERVEUSES CHEZ L'ADULTE: PROGRES RECENTS ET APPORTS DU SYSTEME OLFACTIF

Bauer S.; Patterson P.H.; Moyse E.

S. Bauer, Neurosciences et Systemes Sensoriels, CNRS-UMR 5020; 43, Bld du 11 Novembre 1918, 69622 Villeurbanne Cedex France

Revue de Geriatrie (REV. GERIATR.) (France) 2002, 27/1 (33-44)

CODEN: RGERD ISSN: 0397-7927 DOCUMENT TYPE: Journal; Review LANGUAGE: FRENCH SUMMARY LANGUAGE: ENGLISH; FRENCH NUMBER OF REFERENCES: 61

Neural stem cells that have been demonstrated, since 1992, ensure in vivo localized and adjustable *neurogenesis* in the brain of adult mammals. This discovery brought about a novel concept in neurosciences and great expectations for therapeutics. However, potential use of adult *neurogenesis* to cure or prevent human neurodegenerative diseases demands preliminary answers to several biological issues. The most important one concerns the mechanisms controlling proliferation rate and...
DRUG DESCRIPTORS:

...endogenous compound--ec; epidermal growth factor--endogenous compound --ec; fibroblast growth factor 2--endogenous compound--ec; nerve cell adhesion molecule--endogenous compound--ec; brain derived *neurotrophic* *factor*--endogenous compound--ec; somatomedin C--endogenous compound--ec; transforming growth factor beta2--endogenous compound--ec; interleukin lbeta--endogenous compound--ec; interleukin 6--endogenous compound--ec... MEDICAL DESCRIPTORS:

nervous system development; regeneration; nerve cell plasticity; smelling; degenerative disease; human; nonhuman; *review*

CAS REGISTRY NO.: 62229-50-9 (epidermal growth factor); 218441-99-7 (brain derived *neurotrophic* *factor*); 67763-96-6 (somatomedin C); 9061-61-4 (nerve growth factor)

14/3,K/9 (Item 3 from file: 73)

DIALOG(R) File 73: EMBASE

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11493484 EMBASE No: 2002065231

Neuronal death mode switch and *neurogenesis* as in vivo neuroprotection U ueda H.; H amabe W.

H. Ueda, Department of Molecular Pharmacology, Nagasaki Univ. Sch.

Pharmaceut. Sci., Nagasaki 852-8521 Japan

AUTHOR EMAIL: ueda@net.nagasaki-u.ac.jp

Folia Pharmacologica Japonica (FOLIA PHARMACOL. JPN.) (Japan) 2002, 119/2 (79-88)

CODEN: NYKZA ISSN: 0015-5691 DOCUMENT TYPE: Journal; Review

LANGUAGE: JAPANESE SUMMARY LANGUAGE: ENGLISH; JAPANESE

NUMBER OF REFERENCES: 50

Neuronal death mode switch and *neurogenesis* as in vivo neuroprotection

The brain has various in vivo neuroprotective mechanisms that allow it to survive for an entire lifetime. As well as *neurotrophic* *factor*-mediated inhibition of in vivo apoptotic mechanisms through various protein kinases including Akt and MAP kinase, we propose adding the neuronal death mode switch mechanism...

...to retinal ischemic injury. These findings suggest the possibility that ischemia-induced neuronal death could be inhibited by some drugs to elevate cellular ATP levels. *Neurogenesis* in the adult brain is now an important topic in neuroscience. As brain injury is reported to enhance the *neurogenesis*, this might be also included in the ways of in vivo neuroprotection. As lysophosphatidic acid has various activities to drive *neurogenesis*, the *neurogenesis* could also be managed by other drugs to compensate for functions lost by neuronal death.
MEDICAL DESCRIPTORS:

brain ischemia--etiology--et; neuroscience; brain injury--etiology--et;
neuropathology; apoptosis; cell line; *review*

14/3,K/10 (Item 4 from file: 73)

DIALOG(R)File 73:EMBASE

(c) 2002 Elsevier Science B.V. All rts. reserv.

11441688 EMBASE No: 2002013750

Regulation of adult *neurogenesis* by antidepressant treatment

Duman R.S.; Nakagawa S.; Malberg J.

Dr. R.S. Duman, Abraham Ribicoff Research Facilities, Yale University School of Medicine, 34 Park Street, New Haven, CT 06508 United States AUTHOR EMAIL: ronald.duman@yale.edu

Neuropsychopharmacology (NEUROPSYCHOPHARMACOLOGY) (United States) 2001, 25/6 (836-844)

CODEN: NEROE ISSN: 0893-133X

PUBLISHER ITEM IDENTIFIER: S0893133X0100358X

DOCUMENT TYPE: Journal ; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 42

Regulation of adult *neurogenesis* by antidepressant treatment

Demonstration of *neurogenesis* in adult brain represents a major advance in our understanding of the cellular mechanisms underlying neuronal remodeling and complex behavior. Recent studies from our laboratory and others demonstrate that chronic administration of an antidepressant, including either a 5-HT or norepinephrine selective reuptake inhibitor, up-regulates *neurogenesis* in adult rodent hippocampus. Up-regulation of *neurogenesis* could block or reverse the effects of stress on hippocampal neurons, which include down-regulation of *neurogenesis*, as well as atrophy. The possibility that the cAMP signal transduction cascade contributes to the regulation of *neurogenesis* by antidepressants is supported by previous studies and by recent work. Although additional studies must be conducted to determine the significance of adult *neurogenesis* in humans, these findings will stimulate new avenues of research to identify the cellular and molecular basis of stress-related mood disorders as well as...

DRUG DESCRIPTORS:

cyclic AMP--endogenous compound--ec; monoamine oxidase inhibitor --pharmacology--pd; lithium--pharmacology--pd; morphine--pharmacology--pd; haloperidol--pharmacology--pd; brain derived *neurotrophic* *factor* --endogenous compound--ec; *neurotrophic* *factor*--endogenous compound--ec ; serotonin--endogenous compound--ec; cyclic AMP responsive element binding protein--endogenous compound--ec; rolipram--pharmacology--pd MEDICAL DESCRIPTORS:

... region; stress; down regulation; atrophy; signal transduction; long term exposure; nerve cell; affective neurosis--etiology--et; neuropathology; cell maturation; protein localization; protein expression; human; nonhuman; *review*; priority journal

...CAS REGISTRY NO.: 57-27-2 (morphine); 52-86-8 (haloperidol); 218441-99-7 (brain derived *neurotrophic* *factor*); 50-67-9 (serotonin); 130428-87-4...

14/3,K/11 (Item 5 from file: 73)

DIALOG(R) File 73: EMBASE

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11441687 EMBASE No: 2002013749

Manipulation of neural precursors in situ: Induction of *neurogenesis* in the neocortex of adult mice

Magavi S.S.; Macklis J.D.

J.D. Macklis, Division of Neuroscience, Children's Hospital, Harvard Medical School, 320 Longwood Ave., Boston, MA 02115 United States

AUTHOR EMAIL: jeffrey.macklis@tch.harvard.edu

Neuropsychopharmacology (NEUROPSYCHOPHARMACOLOGY) (United States) 2001, 25/6 (816-835)

CODEN: NEROE

ISSN: 0893-133X

PUBLISHER ITEM IDENTIFIER: S0893133X01003578

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 137

Manipulation of neural precursors in situ: Induction of *neurogenesis* in the neocortex of adult mice

...incapable of generating new neurons. However, in the last decade, the development of new techniques has resulted in an explosion of new research showing that *neurogenesis*, the birth of new neurons, normally occurs in two limited and specific regions of the adult mammalian brain, and that there are significant numbers of...

...precursors in many parts of the adult mammalian brain (Palmer et al. 1995). Recent findings from our lab demonstrate that it is possible to induce *neurogenesis* de novo in the adult mammalian brain, particularly in the neocortex where it does not normally occur, and that it may become possible to manipulate...

DRUG DESCRIPTORS:

somatomedin--endogenous compound--ec; brain derived *neurotrophic* *factor* --endogenous compound--ec; transforming growth factor alpha--endogenous compound--ec; epidermal growth factor; basic fibroblast growth factor MEDICAL DESCRIPTORS:

...cell lesion; olfactory bulb; olfactory epithelium; sensory nerve cell; hippocampus; nerve cell necrosis; brain cortex; vertebrate; protein expression; signal transduction; apoptosis; cell survival; human; nonhuman; *review*; priority journal

CAS REGISTRY NO.: 218441-99-7 (brain derived *neurotrophic* *factor*); 62229-50-9 (epidermal growth factor); 106096-93-9 (basic fibroblast growth factor)

14/3,K/12 (Item 6 from file: 73)

DIALOG(R) File 73: EMBASE

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11327036 EMBASE No: 2001340287

Brain-derived *neurotrophic* *factor* and TrkB tyrosine kinase receptor gene expression in zebrafish embryo and larva

Lum T.; Huynh G.; Heinrich G.

G. Heinrich, Medical Service, Northern California, Health Care System, 150 Muir Road, Martinez, CA 94553 United States

AUTHOR EMAIL: gheinrich@ucdavis.edu

International Journal of Developmental Neuroscience (INT. J. DEV.

NEUROSCI.) (United Kingdom) 2001, 19/6 (569-587)

CODEN: IJDND ISSN: 0736-5748

PUBLISHER ITEM IDENTIFIER: S0736574801000417

DOCUMENT TYPE: Journal ; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 109

Brain-derived *neurotrophic* *factor* and TrkB tyrosine kinase receptor gene expression in zebrafish embryo and larva

The genes that encode the *neurotrophin* family of secreted polypeptides and the Trk family of high affinity *neurotrophin* transmembrane protein tyrosine kinase receptors are induced at the time of *neurogenesis* in mammals and are known to play critical roles in nervous system development. We show here that in contrast to mammals, the genes encoding the *neurotrophin* brain-derived *neurotrophic* *factor* (*BDNF*) and the *neurotrophin* receptor TrkB are expressed throughout embryonic development in the zebrafish. At the embryonic stages preceding transcription of endogenous genes all cells contain *BDNF* transcripts and immunoreactive *BDNF* and the trkB transcripts lack the region that encodes a kinase domain. As development proceeds, progressively fewer cells contain *BDNF* transcripts and by the time of *neurogenesis* the trkB transcripts encode a kinase-domain. In the 4-day-old larva, a small subset of specialized sensory cells on the surface and cells in deeper structures including the gill arches, fin, and cloaca express the *BDNF* gene at high levels in a

promoter-specific fashion. This progressive restriction of *BDNF* gene expression must involve an extinction of *BDNF* gene transcription in some and induction of high levels of transcription in a promoter-specific fashion in other cells. Copyright (c) 2001 ISDN. Elsevier Science... DRUG DESCRIPTORS:

*brain derived *neurotrophic* *factor*--endogenous compound--ec; *tyrosine kinase receptor--endogenous compound--ec; **neurotrophin* receptor--endogenous compound--ec

neurotrophin--endogenous compound--ec; polypeptide--endogenous compound--ec; phosphotransferase--endogenous compound--ec MEDICAL DESCRIPTORS:

...in situ hybridization; immunocytochemistry; reverse transcription polymerase chain reaction; cell culture; Western blotting; cell strain L 929; nonhuman; controlled study; animal tissue; animal cell; embryo; *review*; priority journal

14/3,K/13 (Item 7 from file: 73)

DIALOG(R) File 73: EMBASE

(c) 2002 Elsevier Science B.V. All rts. reserv.

11248847 EMBASE No: 2001263117

Embryonic stem cell-derived *neurogenesis*: Retinoic acid induction and lineage selection of neuronal cells

Guan K.; Chang H.; Rolletschek A.; Wobus A.M.

A.M. Wobus, In Vitro Differentiation Group, Inst. Plant Genet./Crop Plant Res., IPK, Corrensstr. 3, 06466 Gatersleben Germany

AUTHOR EMAIL: wobusam@ipk-gatersleben.de

Cell and Tissue Research (CELL TISSUE RES.) (Germany) 2001, 305/2 (171-176)

CODEN: CTSRC ISSN: 0302-766X DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 57

Embryonic stem cell-derived *neurogenesis*: Retinoic acid induction and lineage selection of neuronal cells

DRUG DESCRIPTORS:

cell receptor--endogenous compound--ec; growth factor; scleroprotein;
neurotrophic *factor*; cytokine
MEDICAL DESCRIPTORS:

cell lineage; nerve cell differentiation; gene expression; protein expression; ion channel; cell survival; dopaminergic nerve cell; human; nonhuman; human cell; animal cell; embryo; *review*; priority journal

14/3,K/14 (Item 8 from file: 73)

DIALOG(R) File 73: EMBASE

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10939981 EMBASE No: 2000430209

Neuroprotective signaling and the aging brain: Take away my food and let me run

Mattson M.P.

M.P. Mattson, Laboratory of Neurosciences, National Institute Aging, Gerontology Research Center, 5600 Nathan Shock Drive, Baltimore, MD 21224-6825 United States

AUTHOR EMAIL: mattsonm@grc.nia.nih.gov

Brain Research (BRAIN RES.) (Netherlands) 15 DEC 2000, 886/1-2 (47-53)

CODEN: BRREA ISSN: 0006-8993

PUBLISHER ITEM IDENTIFIER: S0006899300027906

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 51

^{...} manipulation can increase the brain's capacity for plasticity and

self-repair. Work in other laboratories suggests that physical and intellectual activity can similarly increase *neurotrophic* *factor* production and *neurogenesis*. Collectively, the available data suggest the that dietary restriction, and physical and mental activity, may reduce both the incidence and severity of neurodegenerative disorders in... DRUG DESCRIPTORS:

**neurotrophic* *factor*--endogenous compound--ec MEDICAL DESCRIPTORS:

...Huntington chorea--etiology--et; stroke--etiology--et; calcium homeostasis; calcium cell level; apoptosis; cell regeneration; nervous system development; physical activity; mental performance; mitochondrion; human; nonhuman; *review*; priority journal

14/3,K/15 (Item 9 from file: 73)

DIALOG(R) File 73: EMBASE

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10739228 EMBASE No: 2000219258

Neurotrophic factors in the primary olfactory pathway

Mackay-Sim A .: Meng Inn Chuah

A. Mackay-Sim, Centre for Molecular Neurobiology, Sch. of Biomolecular/Biomedical Sci., Griffith University, Brisbane, QLD 4111 Australia

AUTHOR EMAIL: a.mackay-sim@sct.gu.edu.au

Progress in Neurobiology (PROG. NEUROBIOL.) (United Kingdom) 01 DEC 2000, 62/5 (527-559)

CODEN: PGNBA ISSN: 0301-0082

PUBLISHER ITEM IDENTIFIER: S0301008200000095

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 307

...olfactory pathway, consisting of the olfactory epithelium and olfactory bulb, is presented here as a very useful model for the analysis of growth factor function. *Review* of the available literature suggests that a large proportion of neuroactive growth factors and their receptors are present in the olfactory epithelium or olfactory bulb. Furthermore, the primary olfactory pathway is one of the most plastic in the nervous system with *neurogenesis* continuing to contribute new sensory neurones in the olfactory epithelium and new interneurones in the olfactory bulb throughout adult life. The rich diversity of growth...
DRUG DESCRIPTORS:

**neurotrophic* *factor*--endogenous compound--ec

...protein serine threonine kinase--endogenous compound--ec; Janus kinase
--endogenous compound--ec; STAT protein--endogenous compound--ec; nerve
growth factor--endogenous compound--ec; brain derived *neurotrophic*
factor--endogenous compound--ec; *neurotrophin* 3--endogenous compound
--ec; *neurotrophin* 4--endogenous compound--ec; fibroblast growth factor
--endogenous compound--ec; epidermal growth factor--endogenous compound--ec;
platelet derived growth factor--endogenous compound--ec; somatomedin
--endogenous compound--ec; glial cell line derived *neurotrophic* *factor*
--endogenous compound--ec; transforming growth factor beta--endogenous
compound--ec; scatter factor--endogenous compound--ec; stem cell factor
--endogenous compound--ec; retinoic acid--endogenous compound...
MEDICAL DESCRIPTORS:

paracrine signaling; autocrine effect; olfactory epithelium; olfactory bulb; experimental model; medical literature; sensory nerve cell; interneuron; cell lineage; nerve fiber regeneration; protein family; human; nonhuman; *review*; priority journal

14/3,K/16 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

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10611471 EMBASE No: 2000077541

Neural plasticity to stress and antidepressant treatment

Duman R.S.; Malberg J.; Thome J.

Dr. R.S. Duman, 34 Park Street, New Haven, CT 06508 United States Biological Psychiatry (BIOL. PSYCHIATRY) (United States) 01 NOV 1999, 46/9 (1181-1191)

CODEN: BIPCB ISSN: 0006-3223

PUBLISHER ITEM IDENTIFIER: S0006322399001778

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 55

...to the pathophysiology and treatment of depression. At the cellular level, atrophy and death of stress-vulnerable neurons in the hippocampus, as well as decreased *neurogenesis* of hippocampal neurons, has been reported in preclinical studies. Clinical studies also provide evidence for atrophy and cell death in the hippocampus, as well as... DRUG DESCRIPTORS:

cyclic AMP--endogenous compound--ec; *neurotrophic* *factor*--endogenous compound--ec; glutamic acid--endogenous compound--ec; brain derived *neurotrophic* *factor*--endogenous compound--ec; tianeptine--pharmacology --pd; cyclic AMP responsive element binding protein--endogenous compound --ec; beta adrenergic receptor--endogenous compound--ec; mitogen activated protein kinase...

MEDICAL DESCRIPTORS:

hippocampus; prefrontal cortex; cell death; atrophy; signal transduction; dentate gyrus; brain atrophy; glucose transport; protein expression; cell proliferation; cell differentiation; gene expression regulation; human; nonhuman; *review*; priority journal

14/3,K/17 (Item 11 from file: 73)

DIALOG(R) File 73: EMBASE

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07841302 EMBASE No: 1999084705

Return of the native: Deducing the normal function of the RET proto-

Capes-Davis A.; Robinson B.G.

A. Capes-Davis, Kolling Inst. of Medical Research, Royal North Shore Hospital, St. Leonards, NSW 2065 Australia

Current Opinion in Endocrinology and Diabetes (CURR. OPIN. ENDOCRINOL.

DIABETES) (United States) 1999, 6/1 (61-69)

CODEN: CENDE ISSN: 1068-3097 DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 76

...quest for the normal function of the RET gene, which has moved out of the realms of endocrine neoplasia and into the uncharted waters of *neurogenesis*. This *review* summarizes recent work within this field, commencing with knowledge derived from MEN 2 and moving to an emphasis on the normal function of the RET... DRUG DESCRIPTORS:

glial cell line derived *neurotrophic* *factor*--endogenous compound--ec; neurturin--endogenous compound--ec; neurotropic agent--endogenous compound --ec; mitogen activated protein kinase--endogenous compound--ec; stress activated protein kinase--endogenous compound... MEDICAL DESCRIPTORS:

nervous system development; thyroid medullary carcinoma--etiology--et; familial cancer--etiology--et; signal transduction; embryo development; gene mutation; human; *review*

14/3,K/18 (Item 12 from file: 73)

DIALOG(R)File 73:EMBASE

(c) 2002 Elsevier Science B.V. All rts. reserv.

07488927 EMBASE No: 1998282352

Hirschsprung's disease: A search for etiology

Puri P.; Ohshiro K.; Wester T.

P. Puri, Children's Research Centre, Our Lady's Hosp. for Sick Children,

Crumlin, Dublin 12 Ireland

Seminars in Pediatric Surgery (SEMIN. PEDIATR. SURG.) (United States) 1998, 7/3 (140-147)

CODEN: SPSUE ISSN: 1055-8586 DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 105

...a result of microenvironmental changes after the migration has occurred. Extracellular matrix proteins are recognized as important microenvironmental factors. It has been shown that enteric *neurogenesis* is dependent on extracellular matrices, which provide a migration pathway for neural crest- derived cells and promote the maturation of settled neural crest-derived cells...

...association with some chromosomal abnormalities. Recent expansion of molecular genetics identified multiple susceptibility genes of HD, including the RET gene, the glial cell line- derived *neurotrophic* *factor* gene, the endothelin-B receptor gene, and endothelin-3 gene. Of these, inactivating mutations of the RET gene are the most frequent, occurring in 50...

DRUG DESCRIPTORS:

scleroprotein--endogenous compound--ec; major histocompatibility antigen class 2--endogenous compound--ec; intercellular adhesion molecule 1 --endogenous compound--ec; glial cell line derived *neurotrophic* *factor* --endogenous compound--ec; endothelin b receptor--endogenous compound--ec; endothelin 3--endogenous compound--ec

MEDICAL DESCRIPTORS:

hypothesis; pathogenesis; intestine muscle; disease association; chromosome aberration; molecular genetics; genetic susceptibility; oncogene ret; gene mutation; human; *review*; priority journal

14/3,K/19 (Item 13 from file: 73)

DIALOG(R) File 73: EMBASE

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EMBASE No: 1998263743

Regulation of *neurogenesis* by growth factors and neurotransmitters Cameron H.A.; Hazel T.G.; McKay R.D.G.

H.A. Cameron, Laboratory of Molecular Biology, Natl. Inst. of Neurol.

Dis./Stroke, Bethesda, MD 20892 United States

Journal of Neurobiology (J. NEUROBIOL.) (United States) 1998, 36/2 (287 - 306)

CODEN: JNEUB ISSN: 0022-3034 DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 65

Regulation of *neurogenesis* by growth factors and neurotransmitters

...other factors, including the neuropeptides vasoactive intestinal peptide and pituitary adenylate cyclase-activating peptide, as well as the growth factors platelet-derived growth factor, ciliary *neurotrophic* *factor*, and members of the TGF-beta family, have different effects on proliferation and differentiation depending on the system examined. Expression of many of these factors... DRUG DESCRIPTORS:

...factor alpha; somatomedin c; monoamine; glutamic acid; 4 aminobutyric acid; opiate peptide; vasoactive intestinal polypeptide; hypophysis

adenylate cyclase activating polypeptide; platelet derived growth factor;
ciliary *neurotrophic* *factor*

MEDICAL DESCRIPTORS:

nerve cell differentiation; cell proliferation; germ layer; gene expression; human; nonhuman; embryo; adult; *review*; priority journal

14/3,K/20 (Item 14 from file: 73)

DIALOG(R) File 73: EMBASE

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07385325 EMBASE No: 1998263742

Adult *neurogenesis*: From canaries to the clinic

Goldman S.A.

S.A. Goldman, Dept. of Neurology and Neuroscience, Cornell University Medical College, 1300 York Ave., New York, NY 10021 United States Journal of Neurobiology (J. NEUROBIOL.) (United States) 1998, 36/2 (267-286)

CODEN: JNEUB ISSN: 0022-3034

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 97

Adonis

Adult *neurogenesis*: From canaries to the clinic

...persists throughout much of the songbird forebrain, in mammals it is limited to the olfactory bulb. In humans, the adult SZ appears to largely cease *neurogenesis* in vivo, although it, too, can produce neurons in vitro. In both rats and humans, the differentiation and survival of neurons arising from the postnatal SZ may be regulated by access to postmitotic trophic factors. Indeed, serial application of fibroblast growth factor-2 (FGF-2) and brain-derived *neurotrophic* *factor* (*BDNF*) has allowed the generation and maintenance of neurons from the adult human SZ. This suggests the feasibility of inducing *neurogenesis* in the human brain, both in situ and through implanted progenitors. In this regard, using cell-specific neural promoters coupled to fluorescent reporters, defined progenitor phenotypes may now be isolated by fluorescence-activated cell sorting. Together, these findings give hope that structural brain repair through induced *neurogenesis* and neurogenic implants will soon be a clinical reality.

DRUG DESCRIPTORS:

**neurotrophic* *factor*

fibroblast growth factor 2; brain derived *neurotrophic* *factor*
MEDICAL DESCRIPTORS:

...ventricle; germ layer; ependyma; fowl; telencephalon; olfactory bulb; cell migration; nerve cell differentiation; nerve cell growth; cell specificity; fluorescence activated cell sorter; human; nonhuman; adult; *review*; priority journal

14/3,K/21 (Item 15 from file: 73)

DIALOG(R) File 73: EMBASE

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07232134 EMBASE No: 1998105229

Extrinsic signals in the developing nervous system: The role of neurokines during *neurogenesis*

Heller S.; Ernsberger U.; Rohrer H.

H. Rohrer, Max-Planck-Institute for Brain Res., Department of Neurochemistry, Deutschordenstr. 46, D-60528 Frankfurt/M. Germany Perspectives on Developmental Neurobiology (PERSPECT. DEV. NEUROBIOL.) (United Kingdom) 1996, 4/1 (19-34)

CODEN: PDENE ISSN: 1064-0517

DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 135

Extrinsic signals in the developing nervous system: The role of neurokines during *neurogenesis*

Vertebrate *neurogenesis* involves many distinct differentiation stages that are regulated by extrinsic signals. Survival and differentiation effects on cultured neurons of several lineages are elicited by members of the neurokine family of growth factors, ciliary *neurotrophic* *factor* (CNTF) and the related avian factor, growth promoting activity (GPA). The selective actions of these factors are mediated through the activation of heteromeric receptor complexes...

...localization of CNTFRalpha and GPARalpha is consistent with the previously assigned biological functions but also suggest novel functions for these receptors and their ligands during *neurogenesis*.

DRUG DESCRIPTORS:

*growth factor--endogenous compound--ec; *ciliary *neurotrophic* *factor* --endogenous compound--ec; *growth promotor--endogenous compound--ec MEDICAL DESCRIPTORS:

signal transduction; nerve cell differentiation; cell survival; growth regulation; receptor binding; protein localization; phenotype; nonhuman; animal tissue; animal cell; *review*

14/3,K/22 (Item 16 from file: 73)

DIALOG(R) File 73: EMBASE

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06901650 EMBASE No: 1997186038

NF-kappaB: A crucial transcription factor for glial and neuronal cell function

O'Neill L.A.J.; Kaltschmidt C.

L.A.J. O'Neill, Dept of Biochemistry, Trinity College, Dublin Ireland Trends in Neurosciences (TRENDS NEUROSCI.) (United Kingdom) 1997, 20/6 (252-258)

CODEN: TNSCD ISSN: 0166-2236 DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 64

...An involvement of NF-kappaB in neuronal development is suggested from studies that demonstrate its activation in neurones in certain regions of the brain during *neurogenesis*. Brain-specific activators of NF-kappaB include glutamate (via both AMPA/KA and NMDA receptors) and *neurotrophins*, pointing to an involvement in synaptic plasticity. NF-kappaB can therefore be considered as one of the most important transcription factors characterized in brain to...
DRUG DESCRIPTORS:

n methyl dextro aspartic acid receptor; *neurotrophin*; quisqualic acid receptor; transcription factor--endogenous compound--ec MEDICAL DESCRIPTORS:

alzheimer disease; brain injury; degenerative disease; gene expression; gene expression regulation; inflammation; nerve cell plasticity; nervous system development; priority journal; *review*; signal transduction; synaptic transmission; virus infection; virus replication

14/3,K/23 (Item 17 from file: 73)

DIALOG(R) File 73: EMBASE

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06345524 EMBASE No: 1996006585

Avian olfactory *neurogenesis*

Ayer-Le Lievre C.; Lapointe F.; Leibovici M.

Inst. Embryol. Cell. Molec. UMR 9924, CNRS et College de France, 49 bis, avenue de Belle-Gabrielle,94736 Nogent-sur-Marne Cedex France

Biology of the Cell (BIOL. CELL) (France) 1995, 84/1-2 (25-34)

CODEN: BCELD ISSN: 0248-4900 DOCUMENT TYPE: Journal; Article

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Avian olfactory *neurogenesis*

...is less complex than its mammalian counterpart. Like humans, birds are microsmatic and present the same basic characteristics as other air breathing species. In this *review*, we propose to discuss several aspects of the maturation of the olfactory system during development, paying particular attention to recent advances in this field.

DRUG DESCRIPTORS:

nerve cell adhesion molecule--endogenous compound--ec; nerve growth factor --endogenous compound--ec; neuropeptide--endogenous compound--ec; neurotransmitter--endogenous compound--ec; *neurotrophin*--endogenous compound--ec

14/3,K/24 (Item 18 from file: 73)
DIALOG(R)File 73:EMBASE

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05503149 EMBASE No: 1993271248

iEffects of neurotransmitters and hormones on *neuronal* *production* of *neurotrophins*

Lindholm D.; Castren E.; da Penha Berzaghi M.; Thoenen H.

Department of Neurochemistry, Max Planck Institute for Psychiatry, Am

Klopferspitz 18A, D-8033 Planlegg-Martinsried Germany

Seminars in the Neurosciences (SEMIN. NEUROSCI.) (United Kingdom) 19

5/4 (279-283)

CODEN: SNEUE ISSN: 1044-5765 DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

iEffects of neurotransmitters and hormones on *neuronal* *production* of *neurotrophins*

The *neurotrophins* NGF, *BDNF* and NT-3 are relatively highly expressed by specific populations of neurons in the central nervous system. The levels of NGF and *BDNF* mRNAs in the rat hippocampus are up-regulated by activation of glutamate and muscarinic cholinergic receptors, whereas the gabaergic system exerts an inhibitory influence on these mRNA levels. Glucocorticoid hormones specifically increase NGF and NT-3, but not *BDNF* mRNA levels, in the rat hippocampus. In the rat visual cortex, *BDNF* mRNA, but not NGF mRNA, levels are decreased by light deprivation or after inhibiting the activity of the retinocortical pathway by tetrodotoxin. Exposure to light rapidly elevates *BDNF* mRNA in the visual cortex of dark-reared rats. The rapidity and specificity of changes in *neurotrophin* expression in neurons suggest a possible involvement of these factors in synoptic plasticity during development and in the adult brain.

...pharmacology--pd; dizocilpine--pharmacology--pd; glutamic acid --endogenous compound--ec; kainic acid--pharmacology--pd; messenger rna --endogenous compound--ec; nerve growth factor--endogenous compound--ec; *neurotrophin* 3--endogenous compound--ec; pilocarpine--pharmacology--pd; tetrodotoxin--pharmacology--pd MEDICAL DESCRIPTORS:

animal cell; animal tissue; central nervous system; embryo; gabaergic system; gene expression regulation; hippocampus; light exposure; nerve cell differentiation; nerve cell plasticity; nonhuman; rat; *review*; visual cortex ?ds

Set Items Description
S1 8982 (NEUDOCEMES)

8982 (NEUROGENESIS) OR (NEURONAL (W) PRODUCTION)

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S2
                S1 AND ((NEUROTROPHIC (W) FACTOR) OR (BDNF) OR (NEUROTROPH-
             IN??))
S3
                S2 AND (VECTOR OR (GENE (W) THERAPY))
                RD (unique items)
S4
                S2 AND (VECTOR)
S5
           40
S6
                S2 AND (TREATMENT OR THERAPY)
s7
            0
                S6 AND (NEURODEGENERATIVE (W) CONDITION)
S8
            0
                S6 AND (HUNTINGTON'S (W) DISEASE)
S9
            2
               S6 AND (HUNTINGTON)
S10
            2
                RD (unique items)
           27
S11
                RD S6 (unique items)
                S11 AND REVIEW
S12
           4
S13
           28
                S2 AND REVIEW
           24 RD (unique items)
S14
S15
           1
                S14 AND (HUNTINGTON)
?s (neurodegenerative (w) disease) and (neurotrophic (w) factor)
           25650 NEURODEGENERATIVE
         4449708 DISEASE
            4895 NEURODEGENERATIVE (W) DISEASE
           28507 NEUROTROPHIC
         1791791 FACTOR
           19787 NEUROTROPHIC (W) FACTOR
             167 (NEURODEGENERATIVE (W) DISEASE) AND (NEUROTROPHIC (W)
     S16
                  FACTOR)
?s s16 and (Huntington)
             167 S16
           17544 HUNTINGTON
     S17
              11 S16 AND (HUNTINGTON)
?s s17 and (BDNF)
                 S17
              11
            7897
                  BDNF
     S18
               3 S17 AND (BDNF)
?t s18/3,k/all
 18/3,K/1
              (Item 1 from file: 155)
DIALOG(R) File 155: MEDLINE(R)
08973125
          96335644
                      PMID: 8764649
  Protection of the neostriatum
                                                 excitotoxic
                                        against
                                                                damage
neurotrophin-producing, genetically modified neural stem cells.
 Martinez-Serrano A; Bjorklund A
  Wallenberg
               Neuroscience
                             Center,
                                         Department
                                                      of
                                                           Physiology
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by

and Neuroscience, University of Lund, Sweden.

Journal of neuroscience : the official journal of the Society for Neuroscience (UNITED STATES) Aug 1 1996, 16 (15) p4604-16, ISSN 0270-6474 Journal Code: 8102140

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

Huntington's disease is a progressive *neurodegenerative* *disease* that affects the striatum, above all, the GABAergic striatal projection neurons. In the present study, we have explored the use of genetically modified neural stem cell lines producing nerve growth factor (NGF) or brain-derived *neurotrophic* *factor* (*BDNF*) as a means to protect the striatal neurons against excitotoxic damage after transplantation to the striatum, 1 week before the injection of quinolinic acid into... ... same area. One month after the lesion, striatal degeneration, lesion size, and loss of DARPP-32-positive projection neurons were only slightly affected by the *BDNF*-secreting cells, but substantially prevented when NGF-producing stem cells were used as a source of exogenous trophic factor; innervation of the target fields (pars...

DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.

10517347 BIOSIS NO.: 199699138492

Protection of the neostriatum against excitotoxic damage by neurotrophin-producing, genetically modified neural stem cells.

AUTHOR: Martinez-Serrano Alberto(a); Bjorklund Anders

AUTHOR ADDRESS: (a) Wallenberg Neuroscience Cent., Dep. Physiology

Neuroscience, Univ. Lund, Solvegatan 17, S-223 62**Sweden JØURNAL: Journal of Neuroscience 16 (15):p4604-4616 1996

I(SSN: 0270-6474

D&CUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English

ABSTRACT: *Huntington*'s disease is a progressive *neurodegenerative* *disease* that affects the striatum, above all, the GABAergic striatal projection neurons. In the present study, we have explored the use of genetically modified neural stem cell lines producing nerve growth factor (NGF) or brain-derived *neurotrophic* *factor* (*BDNF*) as a means to protect the striatal neurons against excitotoxic damage after transplantation to the striatum, 1 week before the injection of quinolinic acid into...

...same area. One month after the lesion, striatal degeneration, lesion size, and loss of DARPP-32-positive projection neurons were only slightly affected by the *BDNF*-secreting cells, but substantially prevented when NGF-producing stem cells were used as a source of exogenous trophic factor; innervation of the target fields (pars... MISCELLANEOUS TERMS: BRAIN-DERIVED *NEUROTROPHIC* *FACTOR*; ...

... *HUNTINGTON*'S DISEASE

(4604 - 4616)

18/3,K/3 (Item 1 from file: 73) DIALOG(R) File 73: EMBASE (c) 2002 Elsevier Science B.V. All rts. reserv.

06567598 EMBASE No: 1996229039

Protection of the neostriatum against excitotoxic damage by neurotrophin-producing, genetically modified neural stem cells

Martinez-Serrano A.; Bjorklund A.

Dept. of Physiology and Neuroscience, Wallenberg Neuroscience Center, University of Lund, Solvegatan 17,S-223 62-Lund Sweden Journal of Neuroscience (J. NEUROSCI.) (United States) 1996, 16/15

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LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Huntington's disease is a progressive *neurodegenerative* *disease* that affects the striatum, above all, the GABAergic striatal projection neurons. In the present study, we have explored the use of genetically modified neural stern cell lines producing nerve growth factor (NGF) or brain-derived *neurotrophic* *factor* (*BDNF*) as a means to protect the striatal neurons against excitotoxic damage after transplantation to the striatum, 1 week before the injection of quinolinic acid into...

...same area. One month after the lesion, striatal degeneration, lesion size, and loss of DARPP-32-positive projection neurons were only slightly affected by the *BDNF*-secreting cells, but substantially prevented when NGF-producing stem cells were used as a source of exogenous trophic factor; innervation of the target fields (pars... DRUG DESCRIPTORS:

*brain derived *neurotrophic* *factor*; *excitotoxin--drug toxicity--to; * nerve growth factor; *neurotrophin MEDICAL DESCRIPTORS: